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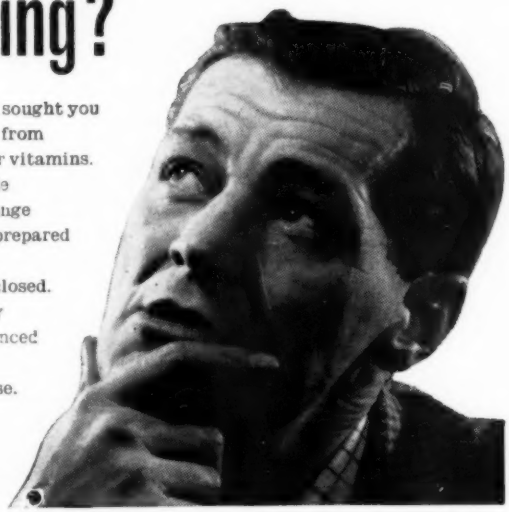

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The Lethal Recessive Problem in Cattle in Guernsey

BY

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Animal Health Division, Ministry of Agriculture, Fisheries and Food, Sittingbourne Road, Maidstone, Kent

SUMMARY.—In this report a brief survey is given on the incidence, identification and steps taken to control the spread of the lethal recessive factor for bulldog calves in the cattle on the Island of Guernsey.

The chance use of two bulls in an artificial insemination scheme that were carriers of this recessive factor demonstrated the extent to which carrier females were present in the cattle population. Fifteen bulldog calves were obtained from 502 calvings from these bulls.

The measures adopted to control its further spread by prohibiting, through Breed Society regulations, the breeding from sons of carriers unless test-mated to show they are clear of the recessive factor, are described and the previous history of this breed defect and the typing of this genetic abnormality are discussed briefly.

History of Abnormality and Early Incidence

IT is now known that the lethal recessive factor, which is responsible for the production of "bulldog" calves, has been present in cattle in the Island of Guernsey for some years. Probably this was initially confined to one or two herds, and in particular the herd in which the casual mutation occurred.

The factor originated by reason of a genetic mutation and not, as is often erroneously believed, by inbreeding. The apparent connexion between the results of inbreeding and the presence of the factor is due to the fact that inbreeding makes the factor more observable and brings to light the heterozygous animals (carriers).

No attention appears to have been paid to the control of the spread of the factor until 1948. In that year subsequent to a visit to the island by the late Dr. A. I. Hagedoorn, a group of enthusiastic breeders formed a "guild," their object being to breed better "Guernseys" within their own herds. Membership consisted of some 18 farmers, 7 of whom were bull breeders, and it is noteworthy that they recognised that the lethal recessive factor was present in the breed. Written statements were obtained from farmers who had seen these abnormal calves in their herds, some useful information was collected, and pedigrees were checked to find the relationships between carrier animals. This showed the necessity for the Breed Society to investigate the problems and at its annual general meeting in 1951 it was resolved that all members should report the birth of bulldog calves and that accurate records should be kept. Although records were kept for some 18 months, no measures of control were introduced and at the end of 1953, 16 bulldog calves had been reported which, together with 7 others recorded prior to 1951, brought the then known total to 23. The seriousness of the problem was demonstrated by the chance use in A.I. during 1957 of 2 carrier bulls which produced 15 bulldog calves out of 502 calvings and positive steps to control it were initiated. It was estimated that 10 per cent. of the animals in the island were carriers.

The problem appeared to be due to a genetic mutation, aggravated by close breeding to an extent understandable in a small island such as Guernsey.

Type of Lethal Factor and Description of Calf

The condition described as a bulldog calf is known scientifically as achondroplasia. This is the term given to a form of dwarfing affecting the bones before birth. Calves are born in a deformed condition, with a marked fore-shortening of the upper and lower jaws, giving the calf an appearance resembling a bulldog ; the limbs are also short (See Fig. 1).

The type of lethal factor, a single mendelian, appears to be similar to that found in the Telemark breed in Norway (Wriedt).

There are several references in the literature to lethal factors in cattle, but the best-known is that of the Dexter breed. In the case of the Dexter the lethal factor is dominant ; when heterozygous it alters normal phenotype and by shortening of limbs it can be readily observed as being present ; when homozygous the factor is fatal. This type of factor is, however, very rare in mammals.

A detailed dissection and histological investigation

of the carcase of a bulldog calf were carried out by Professor Amoroso.

Histological examination of portions of the carcase including reproductive, alimentary and respiratory tracts revealed no abnormality.

Radiographs of the skeleton and cranium, together with measurements which have been compared with the standard linear measurements, confirm that there is some degree of achondroplasia. This is expressed primarily in the changes in the cranium, rather than in skeletal changes (See Figs. 2—7).

The condition appears when the causative recessive gene is homozygous. So far there is no evidence of any abnormality or other characteristic being associated with carriers of the recessive factor (*i.e.* where heterozygous for the gene).

A number of bulldog calves are live at birth but all die within a few minutes. Dystokia as a result of this abnormality is common, and assistance has been necessary in about 40 per cent. of the cases recorded. It is even more serious in the case of first calvers.

A case has recently been recorded where an animal having retained her calf for 3 months after full term was sent for slaughter. *Post-mortem* examination revealed the calf to be a bulldog.

Another case has also been recorded of a cow that calved a bulldog calf and subsequently died as a result of injury during calving, although assistance had been given.

Out of 54 bulldog calves examined each has been found to be identical in type and weight (average 70 lb.), the proportion of males to females being approximately equal.

Incidence and Possible Spread of the Lethal Factor

Carrier to carrier matings on average result in 1 bulldog calf out of every 4 matings. Thus every 4 carrier to carrier matings could result in :—

1 bulldog calf
2 carriers
1 non-carrier (clear of the factor)

In the case of carrier to non-carrier matings, half the progeny on average are carriers. Thus every 2 carrier to non-carrier matings would on average result in :—

1 carrier
1 non-carrier

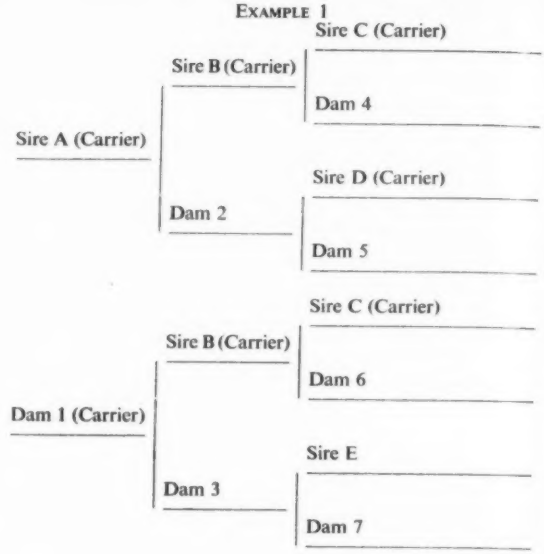
The abnormality, which occurs most frequently in certain blood lines, can usually be traced back to a common ancestor, as shown in the following detailed pedigrees :—

Pedigrees of bulldog calves born.....
In the herds of.....

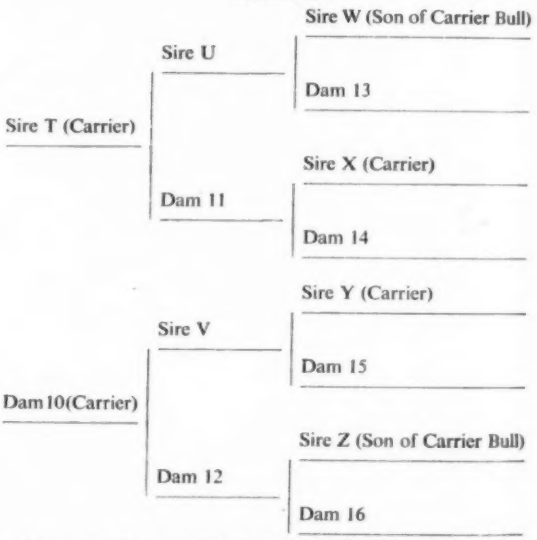
There are only 2 ways in which the factor can be introduced into a clean herd, *viz.* the use of a carrier bull or by purchasing carrier animals from an affected herd.

The time that will elapse before the presence of the recessive factor is revealed varies widely depending on the size of the herd, the proportion of potential carriers, and whether or not a succession of carrier bulls is used. It has been shown that the factor can be present in a herd for some years before its presence is detected.

EXAMPLE 1



EXAMPLE 2



Note.—It will be noticed that in Example 1 the carriers can be traced through the pedigree, but in Example 2 it cannot be ascertained which way the factor was passed.

A study of 2 herds in which the factor suddenly appeared has been carried out and the findings are set out below. No attempt to trace the origin of the factor has been made but merely the introduction of the factor into the 2 herds.

Herd A.

This herd has a history of 6 bulldog calves from 5 animals. The ancestry of the herd was traced back to 2 animals from which the herd has been built up. Neither of these foundation animals have

FIG. 1.—A bulldog calf weighing 70 lb.

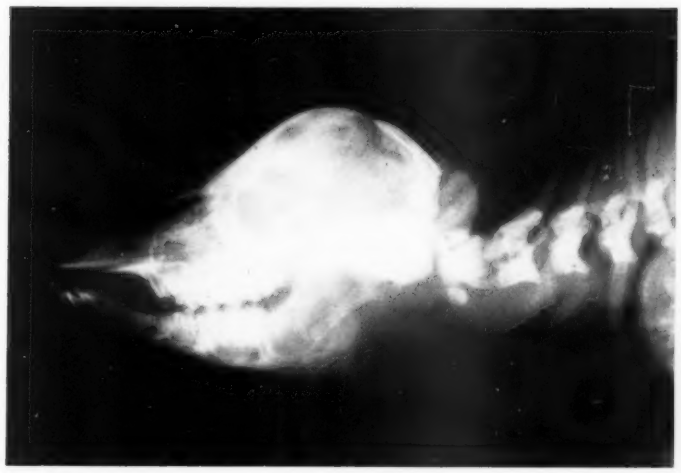


FIG. 2.—Head of a normal calf.

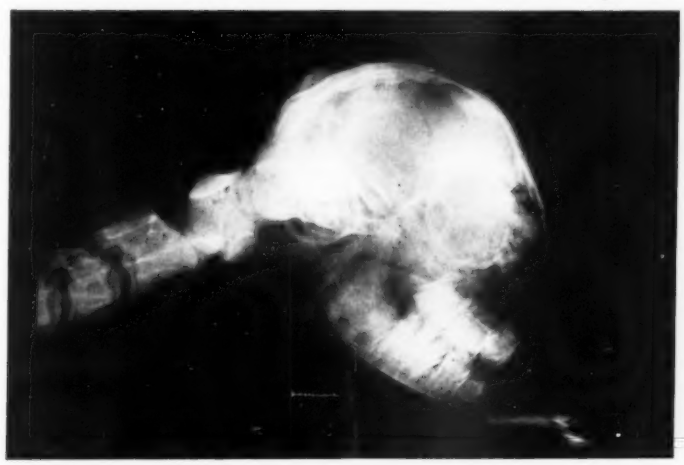


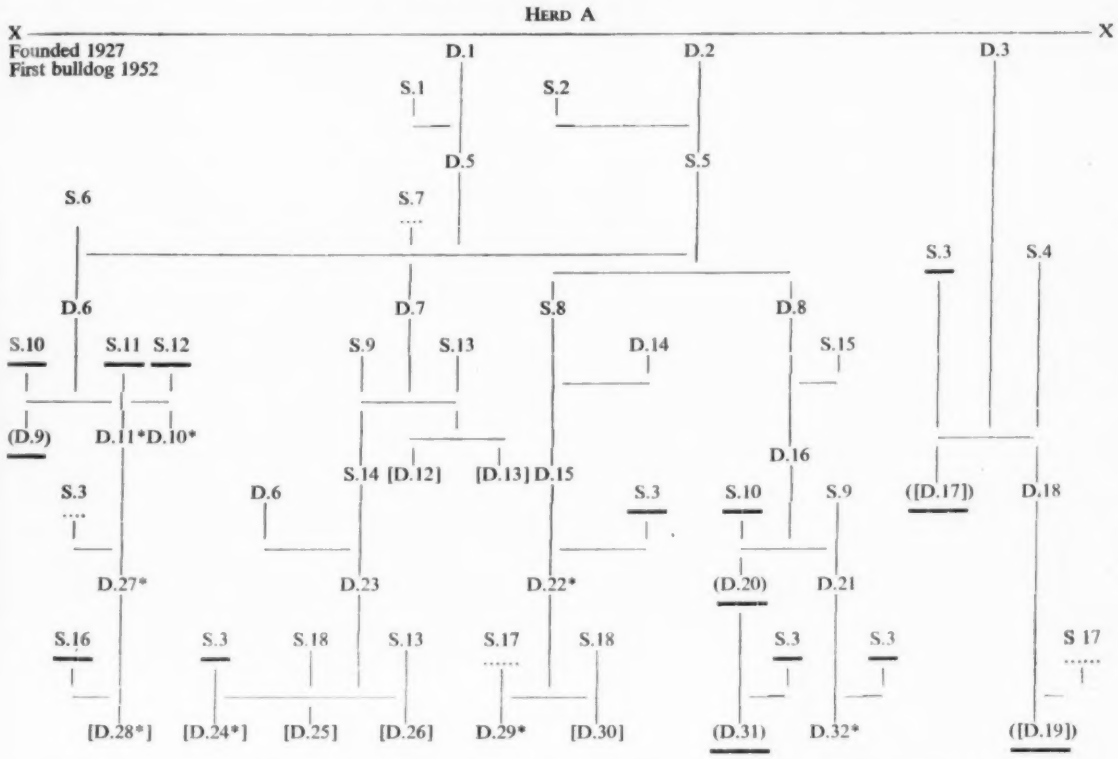
FIG. 3.—Head of a bulldog calf.

FIG. 4.—Normal calf, right fore limb.

FIG. 5.—Bulldog calf, right fore limb.

FIG. 6.—Normal calf, right hind limb.

FIG. 7.—Bulldog calf, right hind limb.



Note.—To avoid complication the coding has been simplified. There is, however, no connection between herds A and B. The herd also contains one other animal which is not shown as its breeding is completely different and it has no trace of bulldogs in its pedigree.

Key.—S = Sire. D = Dam. [ ] = Animals in present herd. ( ) Carrier females which have dropped bulldogs in the herd. — Known carriers. .... Suspected carriers. * Daughter of carrier or son of carrier. X—X Line denoting foundation of herd.

any known trace of lethals in their pedigree and it can safely be claimed that they were clear. The introduction of the factor can be traced to the successive breeding of these animals and their progeny to carrier bulls. At least 5 carrier bulls were used on the herd with the result that the herd at one time contained 5 carriers and 7 daughters of carriers. At present, however, only 2 carriers and 2 daughters of carriers remain in the herd.

Summary

(1) Started with clear stock
(2) Factor bred into herd by use of carrier bulls
(3) Bulldogs resulted from further use of carrier bulls.

Herd B

This is a relatively young herd and the introduction of the factor is mainly due to the purchase of carrier stock. The herd from which these 2 carriers come has no history of bulldogs, the factor being derived from their sires to which the dam of these 2 animals (half sisters) was bred. The effect of this purchase became noticeable when these 2 animals and their 4 progeny were bred to carrier bulls producing between them 4 bulldog calves. The use of carrier bulls has also introduced the factor into the other part of the herd with the result that the herd now contains 4 carriers and 5 daughters of carriers.

Summary

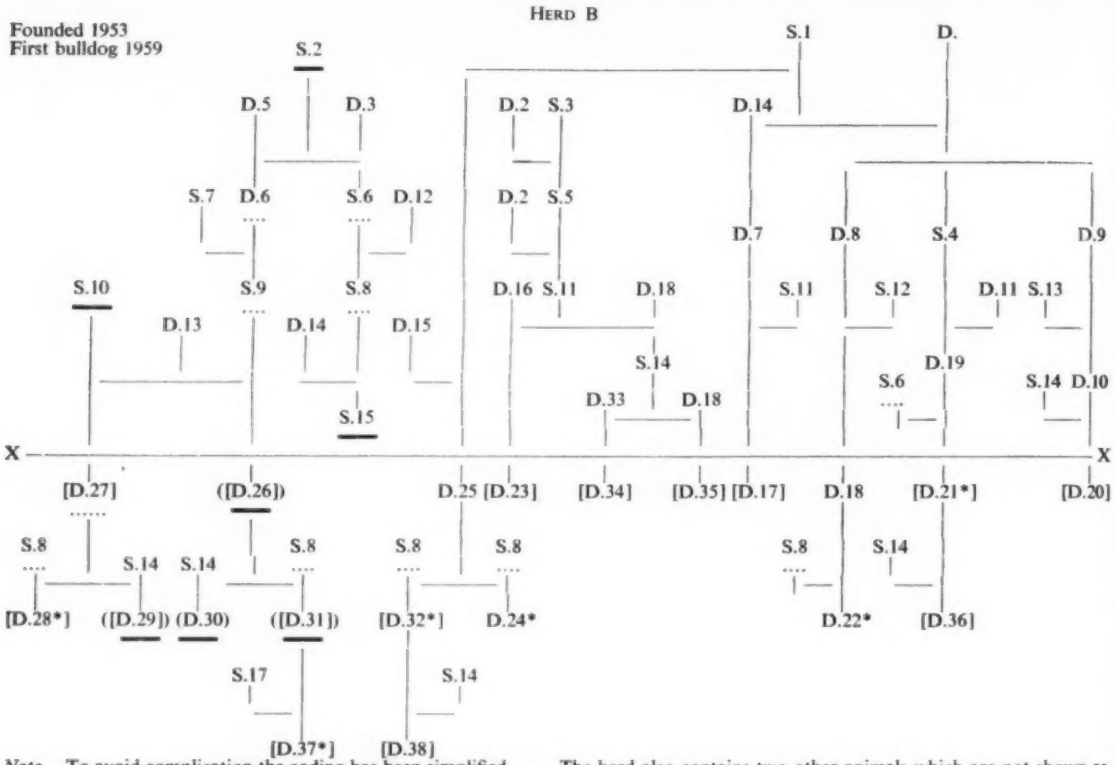
(1) Started with partly carrier stock
(2) Factor bred into herd by use of carrier bulls
(3) Bulldogs resulted in that section bred from the purchased carriers but not so far in the remainder of the herd.

From this analysis it can be seen that when carrier bulls are first used on clear females no effect is apparent. It is only when a succession of carriers is used that the presence of the factor becomes apparent.

Control Measures

I. Test-mating

The fact that the carriers were not identifiable by visual means necessitated test-mating in order to detect the presence of the recessive factor. Wreidt (1930) found that the presence of the factor could be proved by mating a bull with a minimum of 10 known carriers or 20 daughters of known carriers (cows or bulls). If no bulldogs were produced as a



Note.—To avoid complication the coding has been simplified. There is, however, no connection between herds A and B. The herd also contains two other animals which are not shown as their breeding is completely different and they have no trace of bulldogs in their pedigrees.

Key.—S = Sire. D = Dam. [ ] = Animals in present herd. ( ) Carrier females which have dropped bulldogs in the herd. — Known carriers. .... Suspected carriers. * Daughter of carrier or son of carrier. X—X Line denoting foundation of herd.

result of these matings the bull was declared free from the factor. For this reason cows which are known to have produced bulldogs ("carriers") are valuable for test-mating for the presence of the factor. In the same way daughters of carriers are valuable but only half of them are themselves carriers and consequently twice as many are needed to obtain the same degree of coverage.

As a result of this knowledge, test-mating utilising known carriers has been established as a method by which means it can be proved whether or not a bull is a carrier of the factor.

In the light of this knowledge and in pursuance of the agreed policy the 2 carrier bulls were removed from the A.I. scheme and test-mating of A.I. bulls was introduced.

Owners of test-mate animals (carriers or daughters of carriers) were asked to allow these animals to be used for test-mating. These were then mated to the bull undergoing test until the necessary coverage was obtained. At present there are 36 known carriers available for test-mating and approximately 250 daughters of carriers. All test-matings are carried out free and if it is considered necessary, treatment to aid conception, pregnancy diagnosis and assistance at calving are provided without charge.

On average, with the carriers available, it takes 3 months to carry out sufficient inseminations but the results of test-mating are not available until approximately 12 months have elapsed.

Under the first method (Plan 1) a bull is mated with a minimum of 10 carriers and it is also used in the routine inseminations in the island at the same time and the chance mating with animals which are, in fact, carriers but which are not known as such is relied upon to provide a safety margin. In the second method (Plan 2) there is no additional safety margin, such as chance mating, as the bull is used for test-mating only and therefore the bull is mated with a minimum of 8 carriers and 25 daughters of carriers.

This eventually showed that 2 bulls subsequently taken into the A.I. stud were also carriers and they were removed. The results of these test-matings are shown in Table I.

Points to Note :—

(1) It is interesting to note that the majority of bulldogs produced by Bull "B" (6) were from animals in whose lines the factor was not then known to exist.

TABLE I
THE RESULTS OF TEST-MATING UNDER PLAN 1

Bull	Test-matings with known carriers				Chance matings				
					With daughters of known carriers			With unknown	
	Number of animals bred	Normal calves	Bulldog calves	No result	Number of animals bred	Normal calves	Bulldog calves	Number of animals bred	Bulldog calves
"B"	11	5	2	4	8	7	1	141	6
"X"	13	10	–	3	6	6	–	111	–

(2) As will be observed from Table I, the proportion of "bulldog" calves born worked out in practice at the expected rate. Individual exceptions do, however, occur and 3 cases have been recorded where female carriers have produced 2 bulldog calves out of 3 matings, 2 of which were in succession.

II. Inspection of Dead Calves

In 1958 the Breed Society took a further step and made it obligatory for the birth of all calves which were born dead or which died soon after birth to be reported immediately to enable them to be inspected. Since the introduction of this rule 230 calves have been examined and as a result of these inspections 32 bulldogs have been identified.

Blood Typing. Later in 1958 it was suggested that the methods of analysis being used by the Danish Research Laboratory in Copenhagen might succeed in establishing whether there was any association between the blood groups of an animal and its lethal recessive characteristics. Arrangements were made to blood-sample all known carriers, progeny of carrier to carrier matings, and those bulls in the A.I. stud known to be clear from the factor (39 animals in all). However, the workers in Denmark were unable to find any association and test-mating remained the only means of establishing the presence or otherwise of the factor.

III. Control of the Use of Carriers

The Island Breed Society, on the advice of Sir John Hammond, decided that as from April 1st, 1960, no bull calves from male or female known carriers could be registered with the exception that after sons of a carrier had been shown by test-mating to be free from the factor, then the progeny of the bulls would become eligible for registration. Accordingly test-mating facilities through A.I. were offered to all breeders, the cost to be borne by the owner of the bull.

With this decision test-mating under Plan 2, namely to mate a bull with the appropriate number of carriers (or daughters of carriers), was introduced in order to prove the bull to be free from the lethal factor before making the bull available for general use.

Discussion on Result of Control Measures

At first, when no provision for test-mating was made, a bull had to be used extensively before any appreciable chance of throwing a bulldog calf could exist. This was due entirely to the fact that the number of random matings with carriers or daughters of carriers was bound to be relatively few. As a result a number of progeny were retained from bulls which subsequently proved to be carriers and this did little to improve the position (See Table II below).

When test-mating was first introduced (Plan 1) the bull was put into general use but steps were taken to mate the bull as soon as possible with the minimum 10 carriers, thereby reducing the time which elapsed before it was proven clear or otherwise. Plan 1 was acceptable in cases where the possibility of the bull being a carrier was remote, but for sons of carrier bulls its use was not considered advisable. In view of this the more rigorous form of control (Plan 2) was implemented under which the bull is used for test-mating only and not made available for general use until the results of test-mating show the bull to be clear.

It has been shown that by adopting a realistic and positive approach the problem can be solved by

TABLE III
INTERVAL BETWEEN FIRST MATING AND APPEARANCE OF BULLDOG

Bull "A"	Interval 305 days
,, "B"	,, 289 ,,
,, "C"	,, 288 ,,

N.B.—This comparison seems to show that if the factor is present it will appear quickly.

TABLE II
METHODS OF PROVING AND EFFECT OF USE

Method of coverage	Duration of test period (months)	Number of animals bred	Number of calvings	Daughters retained	Sons retained	Bull-dogs
"A" No controlled mating ...	13	330	314	75	12	13
"B" Plan 1	10	160	157	40	1	8
"C" Plan 2	3	49 only	47	8	—	6

N.B.—"A" and "B" and "C" proved to be carriers.

A Modified Rumen Cannula for Cattle

BY

F. A. HARRISON*

Agricultural Research Council, Institute of Animal Physiology, Babraham, Cambridge

SUMMARY.—A modified rumen cannula is described which facilitates the rapid withdrawal of samples of digesta from the rumen. A two-stage operative technique for the insertion of the cannula in cattle is outlined.

THIS paper describes a modification of the rumen cannula designed by Balch and Johnson (1948) which has greatly facilitated the frequent and rapid removal of rumen contents from cattle.

Construction

An exploded photograph of the modified cannula is shown in Plate I and working diagrams in Figs. 1 to 5.

Rubber cannula. This part, which is made by the Avon Rubber Co., is formed as a tube (2 inches deep, 5 inches i.d.) with a wide circular flange (11 inches o.d.) at the lower end and a smaller one (8 inches o.d.) at the upper end. A flap (8 inches o.d.), pierced by 12 equally spaced eyelet-holes, covers the upper flange and is inserted at the inner circumference of the tube. A brass ring (16 S.W.G., $6\frac{1}{8}$ inches i.d. $7\frac{1}{4}$ inches o.d.) with tapped holes (thread 2B.A.) corresponding in position to the eyelets, lies between the flap and the flange. A rubber ring (3/16 inch d.) is inserted on the inside of the tube, $\frac{1}{8}$ inch from the lower end, and acts as a cushion to the plastic plug (Figs. 1 and 2).

Plastic plug. A piece of acrylic tube ("Evolite," 2 inches long, $4\frac{1}{2}$ inches i.d., 5 inches o.d.) is machined to slide easily into the rubber tube. It has a circular flange of acrylic sheet ("Perspex," $\frac{1}{4}$ inch thick, $7\frac{1}{4}$ inches o.d.) at its upper end in which 12 holes (3/16 inch d.) are drilled to correspond in position to the eyelets in the rubber flap. A ring of perspex ($\frac{1}{2}$ inch thick, 4 inches i.d., $4\frac{1}{2}$ inches o.d.) is attached inside the lower end of the tube and the inner surface is coarsely screw-cut (8 threads to 1 inch) to receive a threaded perspex cap. A washer, made from $\frac{1}{4}$ inch plastic belting, ensures a water-tight junction between the cap and the tube (Figs. 3 and 4). To assemble the complete cannula, the plastic plug is inserted into the rubber tube and the two are screwed together by 12 plated countersunk screws ($\frac{1}{2}$ inch by 2B.A.) which pass through the holes in the perspex flange, the eyelet-holes in the rubber flap, and enter the holes in the brass ring (Fig. 5). Compression of the rubber flap between the perspex flange and the brass ring provides a water-tight seal.

Operative Technique

At a preliminary operation a hernia is produced by suturing the rumen wall to the skin and, at a second operation 3 to 4 weeks later, a suitable area of skin plus rumen wall is removed and the cannula inserted. In both procedures the animal is fasted for 24 hours and a drench of chloral hydrate (3-6 g./50 kg. bodyweight) is given 1 to $1\frac{1}{2}$ hours before operation. Paravertebral nerve block (Wright, 1957) is used to anaesthetise the operative area.

1st stage operation. A vertical skin incision, which commences just below the lumbar muscles and passes downwards for 5 inches, is made approximately mid-way between the last rib and the external angle of the ilium. The muscle layers are divided and retracted and the peritoneum is incised. A circular area of rumen wall (5 inches d.) is brought into the incision and sutured to the subcutaneous tissues using interrupted sutures of chromic catgut (No. 4). The skin incision is closed with interrupted sutures of braided nylon (No. 4) (Plate II).

2nd stage operation. A circular piece of skin and adherent rumen wall (4 inches d.) is excised and small bleeding points are tied off with chromic catgut

* Aleen Cust Fellow, Royal College of Veterinary Surgeons. This work was begun during the tenure of an Animal Health Trust Research Training Scholarship.

The Lethal Recessive Problem in Cattle in Guernsey. —*Concluded.*

control of spread through the bulls. If care is taken to ensure that no carrier bulls are used in future, the proportion of carriers born each year will gradually decrease and in a matter of a few generations the problem will be reduced to one of insignificant proportions.

Acknowledgments.—I am very grateful for the assistance which I have received in the preparation of this paper from Sir John Hammond, of the Cambridge School of Agriculture; Professor E. C. Amoroso and Professor J. A. Laing of the Royal Veterinary College, London; Mr. D. R. Melrose of the Ministry of Agriculture, Fisheries & Food, Cattle Breeding Centre, Reading; I am also indebted to Mr. P. H. Blampied, the States of Guernsey Veterinary Officer, to the Royal Guernsey Agricultural and Horticultural Society, to the States Committee for Agriculture and Fisheries for their permission to publish this paper and to the farmers of the island for their assistance.

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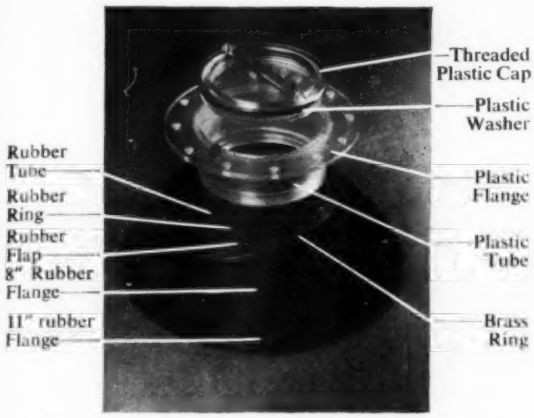

PLATE I.—Exploded photograph of modified cannula.

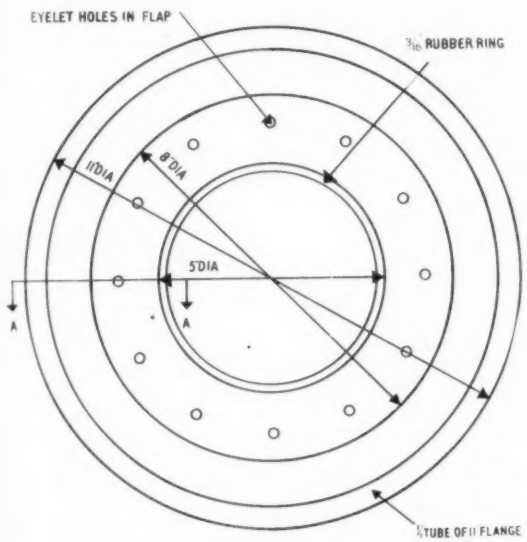

FIG. 1.—Plan of rubber cannula.

(No. 0). The rubber cannula is then inserted and, after ensuring that the inner flange is correctly placed, the plastic plug is pushed into position and screwed down. The perspex surface adjacent to the rubber cannula is smeared with silicone-grease to facilitate subsequent removal of the plug for cleaning purposes (Plate III).

Four steers (12 to 18 months old) have now been fistulated and fitted with the modified cannula, 2 of them over 12 months ago. Post-operative recovery has been satisfactory and normal growth and body-weight increase have been maintained. By the use of this cannula the rumen fistula can be closed efficiently and frequent sampling of rumen ingesta causes little inconvenience to the experimental animal.

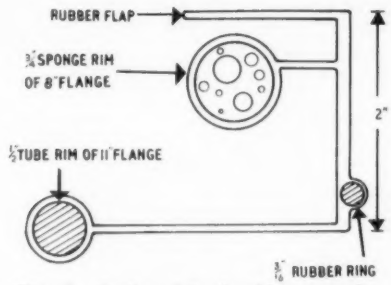

FIG. 2.—Section AA of rubber cannula.

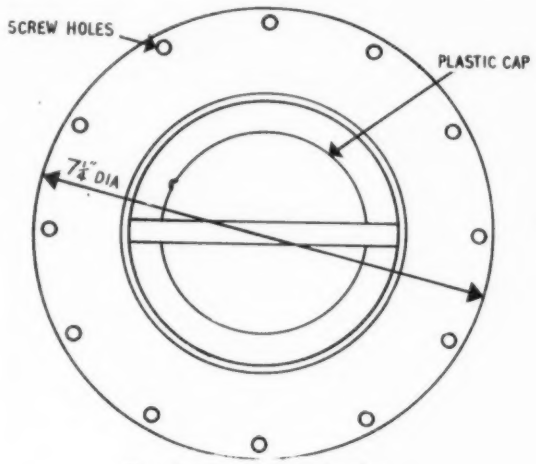

FIG. 3.—Plan of plastic plug.

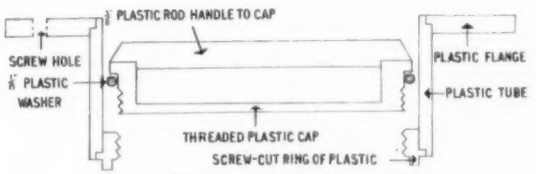

FIG. 4.—Section through plastic plug.

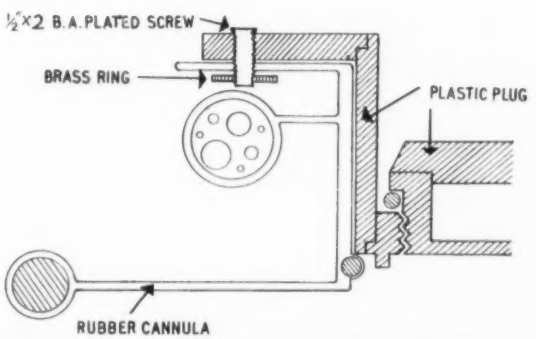

FIG. 5.—Section to show assembly of complete cannula.

An Appraisal of the Value, and Method of Use, of Living Attenuated Canine Distemper Vaccines

BY

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HAIG'S discovery (1948a, b) that distemper virus will grow on the chorio-allantoic membrane (CAM) of embryonated eggs marked the beginning of a new era in the prevention and control of canine distemper (C.D.). It seems reasonable to believe that the decline in the incidence of this disease in Great Britain is not unconnected with the widespread use of living attenuated egg-adapted vaccines.

Haig's virus is usually known as the "Onderstepoort strain," and its origin and history are of some interest. In 1939, Green reported the isolation, and passage through ferrets, of a field strain of C.D. virus. When returned to the dog it was found that substantial attenuation had occurred and the ferret-adapted virus was labelled "distemperoid." Although still causing a proportion of severe reactions, and even some fatalities, "distemperoid" vaccine was widely used and is still favoured by some practitioners in America. Haig further attenuated "distemperoid" virus by adaptation to the CAM of fertile eggs and found that a safe and effective living vaccine could be prepared from infected membranes. The "Onderstepoort" strain has been widely disseminated and is the active agent in many egg-adapted vaccines. Vaccine made with this strain was first prepared on a commercial scale in this country in 1953.

Haig's results were soon confirmed by Cabasso and Cox (1949), using their own strain of C.D. virus, commonly referred to as the "Lederle" strain. A third egg-adapted C.D. virus is the Wisconsin FXNO strain (Bolcher, 1951), and, no doubt, there are others used by commercial firms for vaccine production.

Adaptation to the CAM, in addition to enabling the development of safe and efficient vaccines, has also made possible the direct estimation of virus and neutralising antibody in the laboratory. Since egg-adapted C.D. virus causes recognisable lesions on the CAM, the amount of virus in a given volume of material, for example an ampoule of vaccine, may

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A Modified Rumen Cannula for Cattle.—*Concluded.*

Acknowledgments.—I am grateful to Dr. C. C. Balch (N.I.R.D., Shinfield, Reading) for the loan of a cannula of his design; and to Dr. K. J. Hill for helpful advice and also criticism of the manuscript.

My thanks are due to the Avon India Rubber Co., Melksham, Wiltshire, for their co-operation in the construction of the modified rubber cannula; and to the staff of the Institute workshop who constructed the plastic plugs. Materials were obtained from:—

"Evolite": R. Daleman, Ltd., 325/327, Latimer Road, London, W.10.

"Perspex": I.C.I., Plastics Division, Welwyn Garden City, Herts.

Plastic Belting: K. R. Whiston, Bate Mill, New Mills, Stockport.

PLATE II.—Hernia produced at first stage operation.

PLATE III.—Modified cannula in position after second operation.

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be titrated by inoculating the CAM with serial dilutions. After a suitable period of incubation, the membranes are examined and the highest dilution causing specific lesions in at least half the number inoculated is considered as the 50 per cent. end-point. From this may be calculated the number of egg-infecting doses (EID_{50}) contained in the original material. Furthermore, since specific C.D. antibodies neutralise the effects of virus, the antibody content of immune serum may also be titrated in a similar way. In this case serial dilutions of serum are mixed with standard quantities of virus and, after allowing a period of time for neutralisation, the mixtures are inoculated on to the CAM of fertile eggs as before. The highest dilution of serum preventing the appearance of lesions on the CAM of at least 50 per cent. of inoculated eggs is the end-point, or neutralising titre of the serum. From this may be calculated the number of neutralising units (SN_{50}) in a given quantity of undiluted serum.

The ability to estimate the amount of virus in a vaccine, or the presence or absence of antibodies in a dog's serum, has enabled research workers to gain much knowledge on several important aspects of C.D. immunisation, such as the minimum amount of virus, in terms of EID_{50}, necessary to immunise; the relative value of different vaccines according to antibody response; the duration of colostrol immunity and the correct age to immunise puppies; the duration of immunity induced by vaccination; and the immune status of individuals or communities. Furthermore, the potency and stability of vaccines can more readily be standardised according to virus content, viability after storage, and ability to stimulate the production of antibodies in test animals.

These technical advances, the large number of commercial C.D. vaccines now in use and the variety of advice offered as to their proper application, combined with accumulated knowledge and experience in the field, make this an appropriate time to review the potentialities of living attenuated C.D. vaccines, and to reappraise the basic principles governing recommendations for their use.

Preparation of Egg-adapted C.D. Vaccines

The potentialities of egg-adapted C.D. vaccines are more readily appreciated if the basic procedures employed in their preparation are understood. They all contain living C.D. virus, which is notably labile, especially in the fluid state, when subjected to adverse conditions. When dried and kept frozen, the virus remains viable for long periods, but sustained high temperatures may be a serious source of danger (Piercy, 1961a).

Although each manufacturer evolves a particular routine of production, the basic technical procedures used apply to all C.D. vaccines made in eggs. Fertile eggs are inoculated with attenuated virus and, after an appropriate period of incubation, certain infected tissues are removed, homogenised and freeze-dried. Ampoules may be sealed *in vacuo*, or the vacuum replaced with nitrogen. Potency, safety and toxicity tests are carried out in a number of ways. The amount of virus in an ampoule is estimated, as previously described, by titrations on the CAM of fertile eggs. Provided the number of EID_{50} necessary to immunise a dog is known, and this varies greatly according to strain, level of attenuation and method of propagation, a standard can be fixed below which the virus titre of vaccine must not be allowed to fall. Potency may also be assessed in ferrets, which are highly susceptible to C.D., and this has the added advantage that accidental contamination with virulent virus, however unlikely, will be revealed. Freedom from toxic properties is demonstrated by the inoculation of guinea-pigs and mice. The absence of bacterial contamination may be checked by standard bacteriological methods.

The most critical test, however, is the demonstration of potency in the dog and this is more difficult than might be supposed. For example, prior susceptibility must be proved by demonstrating the absence of serum antibodies. Puppies of the right age must be selected for if too young they may have colostrol antibodies, and if too old they may be relatively resistant to challenge. The dose used to challenge vaccinated puppies must be standardised so that unvaccinated controls will react unequivocably, and an adequate number of animals used to allow for the likelihood of individual variation. On the other hand, the development of the egg-neutralisation test, whereby the production of antibodies following vaccination may be studied, has proved a valuable adjunct to the observation of clinical reactions to challenge.

A recent development is the production of attenuated C.D. virus in tissue cultures (Rockborne, 1958; Cabasso, Kiser & Stebbins, 1959; York, Bittle, Burch & Jones, 1960; Sinha, Stewart, Marshall, Haas & Hawksley, 1960; Piercy, 1961b). This is a more economical way of growing large quantities of vaccine virus, under conditions more highly controlled than possible with fertile eggs. Of particular interest are the reports of York *et al.* (1960) and Sinha *et al.* (1960) which claim that tissue culture C.D. virus is a more efficient immunising agent than C.D. virus grown in eggs. The suggestion is made that egg virus is largely bound up with foreign protein, whereas tissue culture virus is relatively free and better able to multiply in the inoculated dog. These findings have not so far been confirmed in experiments conducted at these Laboratories with the Onderstepoort strain of virus (Piercy, 1961b).

Although this article is primarily concerned with living attenuated C.D. vaccines containing egg-adapted virus, since this type of product is almost exclusively used in this country, it is of interest to note that several other types are used elsewhere. In North America, for example, in addition to egg-adapted strains grown in eggs or tissue cultures, both virulent virus, given by the "serum-simultaneous" or "vaccine-virus" methods, and inactivated vaccines are still commonly used. Green's "distemperoid" vaccine is still favoured by some. Certain products contain more than one strain of virus, although there is, as yet, no experimental evidence of immunological variation between strains. The nearest approach to this was reported by MacIntyre, Trevan and Montgomerie (1948), working with

viruses isolated from the form of C.D. which became known as hard pad disease. In the event, however, laboratory experiments and field evidence have not disproved the subsequent view that hyperkeratosis and encephalitis are no more than particular symptoms of C.D. Goret (1950) and Cabasso (1952) have expressed the opinion that properly immunised dogs will prove resistant to all manifestations of C.D. To the extent that no serological difference between strains has been clearly demonstrated, it is considered that this view is essentially correct, but it may be added that variation in virulence of a single type of virus is a well-known phenomenon and there may be occasions when dogs believed to be properly immunised may yet break down when exposed to an outbreak of unusual severity. Although the pathological significance of hyperkeratosis of the pads and encephalitis may continue to attract attention and stimulate discussion, it is felt that the use of the words hard pad disease, inferring a separate immunological entity, should be discontinued.

The Efficiency of Attenuated C.D. Vaccines

Since successful immunisation with living vaccines depends on the multiplication of injected organisms, the two essential prerequisites are a sufficient number of live virus particles in the inoculum and a susceptible host in which they may grow. Providing these two conditions are fulfilled, a very high proportion of dogs may be immunised with a single dose of egg-adapted C.D. vaccine. It must be emphasised, however, that 100 per cent. success cannot be expected with any biological product, even on theoretical grounds, for in a given population of animals there is likely to be a small proportion incapable of a normal response. This phenomenon is not confined to C.D., or indeed to the use of virus vaccines. In experiments carried out by these laboratories (Piercy & Sellers, 1961), of 172 nine-week-old puppies having no demonstrable maternal immunity, which received a single dose of combined vaccine containing living C.D. and canine hepatitis viruses, 167 developed satisfactory levels of C.D. antibody, an immunising efficiency of 97.0 per cent. Of the remainder, 2 developed low-level antibodies which would probably have been sufficient to protect, and 3 did not respond at all. Only 1 of these three remained available for further study. It was twice revaccinated and on each occasion failed again to develop antibody. Nevertheless, when challenged with virulent virus some months later, this animal remained healthy, apart from a brief febrile reaction which may have been associated with the fact that it was on heat at the time.

These findings confirm that living C.D. and hepatitis viruses, dried in the same ampoule, will multiply in the dog without mutual interference. They are also in close agreement with the results of others. York and Burch (1961), for example, report that 173 out of 179 susceptible dogs (96.5 per cent.) responded to a single dose of tissue culture C.D. vaccine. Baker, Chappel and Dale (1960), who conducted a survey of C.D. immunisation methods in Canada, estimate an overall failure of 5 per cent. Baker (1959) considers that vaccination programmes should be aimed at immunising rather more than 95 per cent.

Clearly, the best of C.D. vaccines will not induce a recognisable antibody response in more than 96 to 98 per cent. of cases, for 1 or 2 dogs in every hundred will probably prove refractory. Such animals are not necessarily at risk, however, for it is likely that they possess a resistance independent of demonstrable neutralising antibodies. This view is sustained by the excellent results obtained with egg-adapted C.D. vaccines over the past 8 years, for if immunisation failures were truly in the region of 2 to 3 per cent., the overall number of breakdowns reported would be infinitely greater than they actually are.

Colostral Immunity and the Active Immunisation of Puppies

The majority of puppies in this country acquire antibody through the first few ingestions of colostrum because the dam is immune, either through vaccination or following recovery from natural C.D. The amount acquired is proportional to the antibody level of the dam. Since colostral antibody declines progressively over a period of weeks, it follows that the higher the level of antibody of the dam, the higher will be the level of antibody passed to the litter, and the longer it will take for a point to be reached when vaccination with live virus may be accomplished.

A detailed study of this problem has been made by Gillespie, Baker, Burgher, Robson and Gilman (1958), who examined over 400 dogs having various levels of maternal antibody. These levels were correlated with the reactions of the dogs when exposed to virulent C.D. virus. In this way a level was established which could be considered as representative of the border-line between susceptibility and resistance. In addition, the time taken from birth for colostral antibodies to fall below the border-line was assessed. Since the decline was progressive, the "extinction" time was proportional to the amount of colostral antibody first acquired. Thus, whereas puppies from dams particularly rich in antibody might not respond to vaccination until some 4 months of age, puppies acquiring very low levels of antibody, or none at all, could be immunised during the first few weeks of life. Following this work, the Cornell group constructed a scale, or nomograph, showing the age at which a litter should be vaccinated according to the antibody level of the dam. By this means, the correct age may be predicted before the puppies are even born, provided the dam's serum is examined beforehand. According to Baker (1959), the nomograph method has given more than 95 per cent. success in field trials.

The Cornell findings are an important contribution to our knowledge of the correct age to immunise puppies. It is of interest that they bear direct comparison with the results of similar research on the colostral immunity problem in rinderpest (Brown, 1958a, b) and African horse sickness (Alexander & Mason, 1941). They have led to the generally held view that 9 weeks old is the optimum age to vaccinate puppies. Exceptions to this, in either direc-

tion, can only be recognised with certainty by examining the sera of the bitch, or the puppy, for the presence or absence of antibodies. The serum neutralisation test is expensive since it involves the use of fertile eggs. It is not suggested that many owners in this country would wish to make use of it as a commercial service. In special circumstances, however, for example the export of pedigree puppies, or when it is particularly desirable to check immune status, it is of great value.

Multiple inoculations are commonly advocated in Canada and the United States. Johnston, York, Robinson, Brueckner and Burch (1959), for example, suggest that puppies liable to early infection should be given 6 injections of inactivated vaccine at weekly intervals, followed by one dose of living vaccine. Some recommend a dose of living vaccine at 9 weeks, followed by a second some 5 to 6 weeks later. Others give repeated doses of serum until the age for active immunisation is reached. Baker *et al.* (1960), in the Canadian survey already referred to, report that over half the veterinarians approached used a second dose of vaccine 2 to 6 weeks after the first. In Great Britain, the general recommendation is a single dose at about 9 weeks old. In the light of more recent experimental knowledge, it may be appropriate to review this principle.

It has been stated that a single dose of potent vaccine can cause a satisfactory antibody response in 96 to 98 per cent. of 9-week-old puppies free of colostral immunity. If 9 weeks is accepted as the optimum age to vaccinate, an important point to be considered is the proportion that will not respond owing to persistent colostral antibody. In the experiments previously referred to, whereas there was a 97.0 per cent. response in puppies having no maternal antibody, the response of the whole group of 225 animals was only 84.9 per cent. This result agrees with the findings of York and Burch (1961) who report that of 102 puppies, ranging from 5 to 10 weeks old, with and without passive immunity, 83.4 per cent. developed antibodies following a single injection of tissue culture vaccine. 55 of the dogs, 20 per cent. of which had failed to respond, were given a second dose 3 to 4 weeks later, and the response in these rose to 98.1 per cent. Baker (1959), also, states that only an 85 per cent. success can be expected when 9-week-old puppies are vaccinated irrespective of colostral antibody level.

Clearly, at 9 weeks of age, maternal antibodies may interfere with the development of active immunity in a significant proportion, and in these animals it would obviously be desirable to delay vaccination for a few weeks to allow maternal antibodies to fall to an insignificant level. A general policy of delay, however, places the majority of puppies at risk from about the 9th week onwards. The rational procedure is to identify those puppies still carrying colostral immunity or to forecast the correct age to immunise in advance, by the serum neutralisation test. Failing this, the alternative is to revaccinate all puppies a month or so later, by which time cases of persistent maternal antibody will be rare. Whether or not to advise a second injection is a matter for sober thought for this would presumably mean that some 85 per cent. of owners were obliged to incur additional expense for the sake of the remaining 15 per cent. In practice, however, it would appear that the great majority of puppies not responding, as judged by the development of demonstrable neutralising antibodies, to vaccination at 9 weeks, have their passive resistance converted to active immunity by natural exposure, or pass uneventfully through mild, subclinical C.D. in later life, or escape exposure altogether, the incidence of C.D. now being comparatively low. An explanation of this kind seems not unreasonable in the light of the overall picture of satisfactory results following a single injection at 9 weeks. There does not, therefore, appear to be any compelling reason for the introduction of a general policy of puppy revaccination.

Duration of Immunity

Claims for prolonged immunity are usually based on the premise that vaccinated animals will, from time to time, come into contact with the disease against which they have been protected. The duration of immunity conferred by living vaccines in animals kept in complete isolation is very largely unknown, for adequately controlled experiments of this sort are few. A reliable guide to persistent immunity is the continuing presence of neutralising antibodies. So far as C.D. is concerned, Cabasso (1957) has found that ferrets remain immune for at least 5 years and there is much evidence to show that a proportion of dogs, given a single dose of living egg-adapted vaccine and thereafter maintained in conditions of isolation, remain resistant for at least several years. At these laboratories, for example, significant levels of antibodies were found in bitches vaccinated 3½ years previously and kept in kennels in which there has been no history of distemper. In another examination, of 8 dogs vaccinated 3 to 4 years previously, 6 had antibodies above the level regarded as the border-line between susceptibility and resistance.

There is also, however, substantial evidence to show that in a significant proportion of vaccinated dogs, neutralising antibodies decline to an undesirably low level within 1 year of vaccination. Baker (1959), summarising important work carried out at Cornell University, considers that about one-third of all vaccinated puppies lose their immunity within a year. He recommends that all dogs be vaccinated annually as a routine safety measure. Howell, Ablett, Keeble and Uvarov (1960) report a gradual fall of neutralising antibody during the first 6 months after vaccination and suggest that revaccination at 12 to 18 months of age may be advisable. Baker *et al.* (1960), in their Canadian survey, state "it would appear that 85 per cent. of the veterinarians in this sampling use booster injections at intervals of approximately 12 months for the first 3 to 4 years of the dog's life." They also record that 27 per cent. were not confident that a natural boosting effect would be provided by street exposure but add that a proportion of these veterinary surgeons practised in rural areas.

When living chick-embryo vaccine was introduced to this country 8 years ago, the incidence of C.D. was high, and the chances of a vaccinated dog, leading a normal life, having its immunity reinforced by natural exposure were excellent. Claims for permanent immunity following a single dose of vaccine were at that time well-founded. During the past few years, however, there has been a notable decrease in the incidence of the disease and the question arises whether a significant proportion of vaccinated dogs may now be escaping the beneficial effect of natural exposure. It seems likely that this may be so, as Howell (1961) has recently pointed out, particularly in rural districts or in areas where a high proportion of the canine population has been regularly immunised over a period of years. There is a risk, therefore, that some vaccinated dogs in this country may have lost their immunity and the proportion may increase should the increase of C.D. decline still further. It is recognised that mature dogs are naturally more resistant to C.D. than puppies, but frank cases of the disease do occur in adult dogs and it seems likely that some cases of milder, ill-defined disease in older dogs may be nothing more than atypical manifestations of C.D. in animals with a low level of resistance.

The day may come when C.D. in this country is so insignificant that the risk of older dogs, whose vaccine immunity has declined, contracting the disease becomes remote. At the present time, however, it is believed that there may be a risk in some areas, and in cases of doubt the rational procedure is to submit a blood sample for serological analysis. Should the examination reveal only a low level of antibody, revaccination is advisable.

The value of revaccination in the presence of immunity is not entirely clear. York *et al.* (1960), for example, have recently claimed that the antibody levels of fully immune dogs can be further increased by a second injection of tissue culture C.D. vaccine, given 4 to 6 months after the first. Gillespie *et al.* (1958), Burgher, Baker, Sarkar, Marshall and Gillespie (1958) and Piercy and Sellers (1960), however, all failed to demonstrate any substantial increase of antibody when immune dogs were injected with virulent virus. In more recent work, Piercy and Sellers (1961) revaccinated 28 dogs whose antibody titres ranged between 1:250 and 1:30,000. The titre accepted at these laboratories as the borderline between susceptibility and resistance is 1:100. In only 2 dogs was there an antibody rise, and in both cases the antibody titres prior to revaccination were of a low order (1:250 and 1:640). It is felt, therefore, that the usefulness of booster injections in dogs already immune is still open to question and cannot be truly evaluated until considerably more research has been done. The value of revaccinating dogs whose antibodies have declined to a low level, however, is not in doubt. Although a serum analysis is the most scientific way of judging the need for revaccination, in practice the owner would presumably be obliged to pay a fee for the examination and a further fee should revaccination be advised. The alternative, and the less expensive way to the owner, is simply to have the animal revaccinated if there is reason to doubt its immune status, and it is likely to be exposed to infection. The practitioner is favourably placed to advise what should be done in the light of such local circumstances as the incidence of C.D. in his district, the history of the animal concerned, the risk involved in going to shows and kennels, and other similar hazards.

Vaccination in the Presence of Infection

In common with other living viral vaccines, chick-embryo C.D. vaccines confer protection within 3 or 4 days of injection due to the "interference" phenomenon. This process is distinct from the creation of immunity by the stimulation of antibodies, which usually takes 2 to 3 weeks. It is generally supposed that vaccine virus, during the process of multiplication, occupies susceptible cells, thus blocking the path for virulent field virus to which the animal may subsequently be exposed. Whether or not this view will become modified by the knowledge now being accumulated about interferon (Isaacs & Lindemann, 1957), an anti-viral substance produced by tissues exposed to virus, remains to be seen. At all events, the "interference" phenomenon can be put to good use in outbreaks of viral disease where living vaccines are available. The time-honoured procedure for the protection of animals in immediate danger of exposure has always been the administration of hyper-immune serum. In many circumstances, however, living vaccines can be given for the same purpose. It is questionable whether purely anti-viral serum is effective once clinical symptoms have appeared, for by this time the tissues have been invaded, cell destruction is taking place, and serum cannot neutralise virus inside cells. There is, of course, an application in such cases for serum containing antibodies against the bacteria commonly responsible for secondary symptoms. The immunity provided by serum is only passive, however, and of short duration, commonly necessitating a subsequent inoculation of vaccine. The circumstances in which anti-viral serum can be effective against C.D. are, therefore, limited. Its value when given shortly before exposure is not in doubt, but if a dog has not yet been exposed, but is at risk, attenuated vaccine will provide rapid protection and, moreover, confer active immunity. In this connexion, experiments carried out by Baker, Leader and Gorham (1952) and Cabasso, Stebbins and Cox (1953), showed that chick-embryo vaccine given to ferrets 2 days before virulent virus prevented the development of distemper. Wilde and Scott (1961) have reported a similar phenomenon in cattle given attenuated rinderpest vaccine 2 days before challenge with virulent rinderpest virus.

From time to time field evidence is offered in support of the view that chick-embryo vaccine, given to established cases of C.D., has a therapeutic effect. It is hard to believe that any such benefit can be other than non-specific in character. Since only one serological type of C.D. virus has been recognised, it is difficult to understand how an attenuated virus could multiply and exert some specific effect in an

(*Concluded at foot of p.* 949)

News and Comment

RABIES IN AFRICA

Hundreds of people are being treated against rabies in hospitals in Ruanda Urundi, where a serious outbreak of the disease has just been encountered, reports the Animal Health Branch of FAO. "The virus could be harboured among wild animals such as skunks, jackals, leopards, wild cats, foxes and even, we now know, in bats," the report continues. "If wild animals were to become infected the situation would get completely out of control."

The danger of an epidemic exists because of the shortage of veterinarians, doctors and medical assistance in that part of Africa.

For a long time it has been known that carnivorous vampire bats are symptomless carriers, transmitting the disease to animal and man. But, since 1951, evidence has shown that insectivorous bats also carry and transmit the disease. Several cases of rabies transmitted by such bats, for long considered harmless and even useful animals, have been diagnosed in the United States. Some of these cases were fatal because neither the person bitten nor the doctors concerned know that bats could be rabid.

The seriousness of the situation is indicated by the fact that bats are the most numerous mammals on earth. They are known practically all over the world, living by their millions in caves, caverns and forests, which makes their control extremely difficult. It is also known that certain species of bats migrate over long distances, which again considerably increases the danger of contagion. The report concludes:—

"World-wide co-operation in tackling this new problem is indispensable, but this can be successful only if it is supported by the public in all countries. People must be made aware of this new danger, report on any abnormal behaviour of bats and, of course, seek immediate medical attention if anyone is bitten."

THE ROYAL VETERINARY COLLEGE ASSOCIATION

We are indebted to Professor McCunn for the following note.

On September 7th last the members of this Association met at dinner in one of the most ancient Halls in Oxford—Saint Edmund Hall. Its name commemorates Edmund of Abingdon (d.1240), the saint and scholar who was the first member of the University to become Archbishop of Canterbury and to be canonised. This ancient Hall succeeded in maintaining its independent character through all the vicissitudes of time and in 1937 the Queen granted it a charter of incorporation with all the rights and privileges of a full college. What better venue could there be for the graduates of the premier veterinary college of Great Britain than that they too should be privileged to spend a convivial evening together in the lovely precincts of the buildings which lodged the earliest form of residential society for undergraduates in Oxford.

After the loyal toast had been honoured and the traditional toast "To the Royal Veterinary College

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An Appraisal of the Value, and Method of Use, of Living Attenuated Canine Distemper Vaccines.—*Concluded.*

animal already infected with virulent virus. Perhaps a comment by Hudson (1955), who pointed out that injections of normal horse serum or condensed milk sometimes apparently influence the outcome, is not irrelevant. It is also of interest that Scott (1961) was unable to influence the course of rinderpest by inoculating attenuated caprinised virus into cattle already infected with the disease.

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the premier College" had been received with acclamation, the President gave a short account of the state of the Royal Veterinary College Association. He regretted that owing to the illness of Mrs. Wragg their guest of honour, Captain W. G. Wragg, had not been able to make the journey to Oxford. Captain Wragg had been looking forward to this occasion, when he could meet many old friends again, with eager anticipation. The President felt sure they would want him to write to Captain Wragg, on behalf of the Association, express their regret at his absence, and send their best wishes for Mrs. Wragg's speedy recovery.

The Association was in a healthy state. During the last year many new members had joined. He, personally and by direct contact, had been able to enrol about 130 new members, but even so the association was still very far from its potential membership peak. He asked that all present should try to get at least one new member to join during the coming year. On the financial side, the policy of investing the small life membership fees was already bearing fruit, and they had an income which covered most of their simple expenses for postage, etc. It was his great desire that this income should increase so that they might be able to give positive help to any old "Londoner" who might be in need. This was his vision and dream of the future. If it had been done by the old students of his other Alma Mater, the London Hospital, it could be done by the graduates of the Royal Veterinary College. The health of a society depended much upon its officers, and he took this opportunity of thanking their secretary Kelvin Price and their treasurer Dennis Green together with Keith Barnett who had undertaken the major burden of organising that evening's event.

Mr. Marston, President of the Students' Union, then gave an account of the year's social and sporting activities. He drew particular attention to the Royal Veterinary College East African Expedition. The object of this was to study the veterinary aspects and problems associated with the conservation of animals living in their natural state. This adventure would be worth while for its scientific interest, and he hoped it would bring credit to the Royal Veterinary College. At a later date they might ask their friends for financial support. (The members present responded immediately, and £58 was handed to Mr. Marston.)

UNIVERSITY NEWS

Edinburgh

The undermentioned *external* candidates have passed the First Professional Examination for the degree of Bachelor of Veterinary Medicine and Surgery (B.V.M. & S.) in the subject(s) stated below:—

Physics: Holt, Keith; *Chemistry*: Holt, Keith; *Biology*: Arnold, Ian P.

The undermentioned candidates have passed the First Professional Examination for the B.V.M. & S. degree in the subject(s) stated below:—

Physics: Akafekwa, George I.; Bardsley, Daniel H.; Brown, Morag E.; Burnie, Andrew G.; Cuthbertson, John C.; Davies, Dafydd H.; Downhill, Ian R.; Fell, William L.; Gordon, Irene E.; Hill, Peter T.; Imray, William S.; Jack, Laurence C.; McIntosh, Graham H.; Mackenzie, Gordon S.; Murray, Roy; Ojo, Matthew O.; Orr, William L.; Purvis, Graham M.; and Wekesa, Noah M.

Chemistry: Gordon, Irene E.; Hamilton, Doreen E.; Orr, William L.; and Pringle, John D.

Biology: Barnes, Peter; Cuthbertson, John C.; Dixon, Francis W.; Hally, John; Hothersall, Dennis; Imray, William S.; Jaggar, David H.; Lamont, William N.; Leslie, Fiona M. R.; McIntosh, Graham H.; Orr, James P.; Orr, William L.; Pringle, John D.; Thomson, John; Wekesa, Noah M.; and Welch, Robert R.

London

Two Special University Lectures, of interest to members of the profession are announced, as follows:—

"Myxomatosis: A Study of Evolutionary Changes in an Infectious Disease" by Professor Frank Fenner, M.B.E., M.D., F.R.S., Professor of Microbiology in the Australian National University. This lecture will be delivered at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C.1, at 5.30 p.m. on Friday, November 10th, 1961. The chair will be taken by Professor C. F. Barwell, M.A., M.D., Goldsmiths' Company Professor of Bacteriology in the University of London. Admission is free, without ticket.

"Scrapie: A Transmissible Disease of Sheep" by Dr. J. T. Stamp, M.R.C.V.S., F.R.S.E., Director, Animal Diseases Research Association, Moredun Institute, Edinburgh. The lecture will be delivered at the Royal Veterinary College, Royal College Street, N.W.1, at 4 p.m. on Tuesday, November 14th, 1961. The chair will be taken by Professor R. Lovell, D.SC., PH.D., M.R.C.V.S., D.V.S.M., Professor of Veterinary Bacteriology in the University of London. Admission is free, without ticket.

THE HARKNESS FELLOWSHIPS

Thirty Fellowships, under the patronage of Her Majesty The Queen, are offered in 1962 to candidates from the United Kingdom, under the following conditions:—

Awards are restricted to British subjects who are citizens of the United Kingdom and Colonies by birth or by naturalisation.

Candidacy is open to men and women in any profession or field of study who will be between 21 and 32 years of age on September 1st, 1962. Preference will normally be given to younger candidates. Candidates should by that date have a degree from a university in the United Kingdom or, in the case of category C below, the equivalent in qualifications conferred by professional bodies, or an attested level of professional competence. The training, experience and intention of such candidates should fall within one of the following groups:—

A. Recent graduates or graduands who desire further preparation (in the same or a different field) for non-academic careers. Candidates in this group are expected to spend 21 months on the Fellowship.

B. Men and women who have already had or are in course of post-graduate training in the United Kingdom and wish to continue their studies,

research or investigations in the United States with a view to an academic or professional career in the United Kingdom or a Commonwealth country.

C. Men and women with substantial practical experience in public service, the professions, the creative arts, journalism, branches of business or industry, or other comparable careers.

Selection of Fellows will take into account those qualities of character, intellectual ability, personal aspirations and potential leadership necessary to realise the international purpose of the awards.

The purposes of the Fellowships are best served when a Fellow can spend the equivalent of two consecutive academic years, including the months of required travel, in the United States. In the case of younger Fellows, this 21 months' tenure is regarded as normal.

Fellows are required to follow a definite programme of study or investigation approved by the Fund officers, and are expected—even when their projects call for extended travel—to be based at selected institutions or locations.

After completion of the Fellowship, a Fellow shall render a confidential report or reports on his work and experiences in the United States.

Fellowship emoluments cover expenses of travel, residence and study during tenure, as follows:—

A resident living allowance of $225 a month; book and equipment allowance of $300 payable on arrival in New York; tuition and research fees and expenses; travel from a Fellow's home to the United States and return; American travel allowance, Christmas: $150; American travel allowance, General: $175 a month for 3 months: automobile rental, provided by the Fund normally for 6 months; health insurance. There are, in addition, certain supplementary allowances.

Further information and application forms may be obtained from the Warden of Harkness House, London. Applications, typed and in duplicate, should be submitted on the prescribed forms to reach the Warden of Harkness House, London, at any time *before Wednesday, November 15th, 1961. Applications cannot be accepted after this date.*

PERSONAL

Births

DAWSON.—On September 16th, 1961, to Valerie, wife of Peter Dawson, B.VET.MED., M.R.C.V.S., of Linden Walk, Louth, a daughter, Claire Elizabeth.

DENHAM.—On September 4th, 1961, to Esther Denham, M.R.C.V.S., and Peter Denham of 6, Parkway Close, Welwyn Garden City, Herts., a son, Simon David, brother to John and Sarah.

DEVILE.—On September 16th, 1961, to Cynthia, wife of C. P. DeVile, B.VET.MED., M.R.C.V.S., 35, Arlington Road, Eastbourne, a daughter.

JEFFREY.—On September 6th, 1961, to Marian, wife of Bryan Jeffrey, M.A., VET.M.B. (CANTAB.), M.R.C.V.S., of Langthwaite, Lanercost Road, Brampton, Cumberland, a son, Paul.

RETIREMENT OF MR. R. J. P. WATSON

To mark his retirement from the Animal Health Division of the Ministry of Agriculture, Mr. R. J. P. Watson was entertained to dinner in the Gordon Arms Hotel, Elgin, on September 13th, by his colleagues in practice and in the Ministry. Mr. Watson was presented with a camera and telephoto lens.

Good wishes were sent by telegram from Mr. D. S. Barbour, deputy C.V.O., Edinburgh, and by Sir John Ritchie, C.V.O.

Mr. Watson has been divisional veterinary officer of Banff, Moray and Nairn since 1938.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

MAHMOODULLAH, Mohamed, Ministry of Animal Resources, Khartoum, Sudan. Graduated from the Royal Veterinary College, London, December 21st, 1933. Died at Hyderabad, India, September, 1961.

COMING EVENTS

October

4th (Wed.). WESTERN COUNTIES VETERINARY ASSOCIATION. A meeting of the Devon Branch will be held at the Passage House Inn, Kingsteignton, Nr. Newton Abbot, 7.30 p.m.

NORTH OF SCOTLAND. Autumn Meeting in the Gordon Arms, Elgin, 2.30 p.m.

5th (Thurs.). WEST OF SCOTLAND DIVISION. Meeting at the University Veterinary Hospital, Bearsden Road, Glasgow, 7.30 p.m.

AYRSHIRE VETERINARY ASSOCIATION. Meeting in the Staff Common Room, Agricultural College, Auchincruive, 7.30 p.m.

EAST MIDLANDS DIVISION. Autumn Meeting at the Veterinary Science Division of Boots Pure Drug Co. Ltd., The Priory, Thurgarton, Notts, 1 p.m.

CENTRAL VETERINARY SOCIETY. Meeting at the Royal Veterinary College, Camden Town, London, N.W.1, 6 p.m.

6th (Fri.). MIDLAND COUNTIES VETERINARY ASSOCIATION. Joint meeting with the Shropshire Veterinary Club at the Oak Hotel, Shelton, Shrewsbury, 8.15 p.m.

10th (Tues.). WEST WALES VETERINARY CLUB. Farewell Dinner for Mr. E. I. Williams at the Ivy Bush Hotel, Carmarthen.

11th (Wed.). B.S.A.V.A. KENT REGION. Meeting at the Great Danes Hotel, Ashford Road, Maidstone, Kent, 7.45 p.m.

B.S.A.V.A. MIDLAND REGION. Joint Meeting with the Staffs. and Derby Veterinary Club at the White Hart Hotel, Uttoxeter, 8.15 p.m.

SUSSEX VETERINARY SOCIETY. Meeting at the Old Ship Hotel, Brighton, 2.30 p.m.

EASTERN COUNTIES VETERINARY SOCIETY. Meeting at the Belstead Brook Hotel with a tour of Messrs. R. & W. Paul's mills, 12 noon.

TECHNICAL DEVELOPMENT COMMITTEE OF THE B.V.A. Meeting at 7, Mansfield Street, Portland Place, London, W.1, 10.30 a.m.

12th (Thurs.). B.S.A.V.A. SOUTH WALES REGION. Meeting at the Queens Hotel, Queen Street, Cardiff, 7 p.m.

HERTS. AND BEDS. VETERINARY SOCIETY. Meeting at the George Hotel, Luton, 7.30 p.m.

NORTH WILTS. VETERINARY CLUB. Meeting at the Goddard Arms, Swindon, 7.30 p.m.

MID-WEST VETERINARY ASSOCIATION: CLINICAL DISCUSSION GROUP. A meeting will be held at the Mauretania, Bristol, 7.30 p.m.

17th (Tues.). MID-WEST VETERINARY ASSOCIATION. Supper-Dance in aid of the Victoria Veterinary Benevolent Fund at the Berkeley Café, Clifton, 9 p.m.

18th (Wed.). SOCIETY OF PRACTISING VETERINARY SURGEONS. Joint Meeting with the Lakeland Veterinary Association and the Dumfries and Galloway Division at Siddles' Crown Hotel, King Street, Penrith, Cumberland, 8 p.m.

20th (Fri.). SOUTH WALES DIVISION. Annual Dinner Dance at the Dragon Hotel, Swansea.

SCOTTISH METROPOLITAN DIVISION. Annual Dinner Dance in the Carlton Hotel, North Bridge, Edinburgh, 8.30 p.m.

26th (Thurs.). WESTERN COUNTIES VETERINARY ASSOCIATION. Meeting at the Rougemont Hotel, Exeter, 2.15 p.m.

28th (Sat.). B.S.A.V.A. NORTH WESTERN REGION. Meeting at the Queen's Hotel, Manchester, 7.30 p.m.

November

2nd (Thurs.). R.A.V.C. DIVISION. Meeting at Bristol University Veterinary College.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the date on which disease has been confirmed, followed by the postal address and local authority.

Anthrax

September 20th, 1961. W. Cox, Stockadon Farm, Landulph, Saltash, Cornwall (Parish: Landulph). (Cattle.) CORNWALL.

September 20th, 1961. J. Johnson, Trust Farm, Kelsall, Saxmundham, Suffolk (Parish: Kelsall.) (Cattle.) EAST SUFFOLK.

September 20th, 1961. R. W. G. Caynes, Tredinnick Duloe, Looe, Disease at General Farm, The Glebe, Duloe, Looe, Cornwall (Parish: Duloe). (Cattle.) CORNWALL.

September 21st, 1961. H. F. Peake, Boveway, Liskeard, Cornwall (Parish: Liskeard). (Cattle.) CORNWALL.

September 21st, 1961. D. A. James, High Court Farm, Standerwick, Frome, Somerset (Parish: Beckington). (Cattle.) SOMERSET.

September 22nd, 1961. R. F. Thayer, Church Farm, Hornblotten, Shepton Mallet, Somerset (Parish: Hornblotten). (Cattle.) SOMERSET.

September 22nd, 1961. A. R. Gates, 136, Finedon Road, Irthlingborough, Wellingborough, Northants. Disease at: Big Meadow, Park Farm, Irthlingborough, Wellingborough, Northants (Parish: Irthlingborough). (Cattle.) NORTHAMPTONSHIRE.

September 22nd, 1961. S. J. Mitchell, Jaysland, St. Cleer, Liskeard, Cornwall (Parish: St. Cleer). (Cattle.) CORNWALL.

September 25th, 1961. Geach Bros., Treworrick, St. Cleer, Liskeard, Cornwall (Parish: St. Cleer). (Cattle.) CORNWALL.

September 25th, 1961. J. Young, Auchans Farm, Johnstone, Renfrews. (Parish: Kilbarchan). (Cattle.) RENFREWSHIRE

September 25th, 1961. G. J. Martin, Towell Farm, St. Dominic, Callington, Cornwall (Parish: St. Dominic). (Cattle.) CORNWALL.

Fowl Pest

September 19th, 1961. J. W. Lovick, Breck Farm, Upgate, Swannington, Norwich, Norfolk. NOR 82X. NORFOLK.

September 19th, 1961. Denton Turkey Breeders Ltd., West Hall, Mundford, Thetford, Norfolk. NORFOLK.

September 19th, 1961. R. A. Sabberton, 16, Lous Close, Old Catton, Norwich, Norfolk. NOR 70N. NORFOLK.

September 19th, 1961. R. L. Nice, Heathlands, Thurston, Bury St. Edmunds, Suffolk. WEST SUFFOLK.

September 19th, 1961. C. Betts, Church Farm, Morningthorpe, Long Stratton, Norwich, Norfolk. NOR 72W. NORFOLK.

September 19th, 1961. W. Mussett, The Bungalow, Morningthorpe, Long Stratton, Norwich, Norfolk. NOR 72W. NORFOLK.

September 21st, 1961. S. E. Fuller, Kewsington Villa, Hockley Road, Rayleigh, Essex. ESSEX.

September 21st, 1961. Miss W. Richmond, Old Post Office, Weston Longville, Norwich, Norfolk. NOR 59X. NORFOLK.

September 23rd, 1961. A. H. Johnson, Chartley Turkey Farm, Stowe by Chartley, Stafford. STAFFORDSHIRE.

September 23rd, 1961. Mrs. H. Kubiak, Green Lane, Mundford, Thetford, Norfolk. NORFOLK.

September 24th, 1961. G. F. Garrett, Holme Farm, Seething, Norwich, Norfolk. NOR 35X. NORFOLK.

September 25th, 1961. I. G. Berry, 28, Tyechurst Hill, Loughton, Essex. Disease at: Forest House, Woodside, Thornwood, Epping, Essex. ESSEX.

Swine Fever

September 19th, 1961. A. M. Allen, Pear Orchard Farm, Cam, Dursley, Glos. GLOUCESTERSHIRE.

September 19th, 1961. T. Greenway, 63, Ash Vale, Nantybwch, Tredegar, Mon. Disease at: Quick Pits Farm, Nantybwch, Mon. MONMOUTHSHIRE.

September 19th, 1961. T. Naden, Fields Farm, Ballidon, Ashbourne, Derbys. DERBYSHIRE.

September 20th, 1961. E. L. Stevens, Little Newra Farm, Goldcliffe, Newport, Mons. MONMOUTHSHIRE.

September 20th, 1961. R. S. McCorquodale (Mrs.), Home Farm, Stoke Rochford, Grantham, Lincs. LINCOLNSHIRE (KESTEVEN).

September 20th, 1961. I. Jones, 5, Devon View Terrace, Port Tennant, Swansea. Disease at: Kilvey Hill, Port Tennant. COUNTY BOROUGH OF SWANSEA.

September 20th, 1961. G. A. Hall, Parkside Farm, South Cave, Brough, Yorks. YORKSHIRE, EAST RIDING.

September 20th, 1961. J. W. Spier, Micklewood Cottage, Otherton, Penkridge, Stafford. STAFFORDSHIRE.

September 21st, 1961. J. Shelley, Meaford Old Hall Farm, Meaford, Stone, Staffs. STAFFORDSHIRE.

September 21st, 1961. A. Ll. Davies, Dinas Isaf Farm, Edmunston, Tonypandy, Glamorgan. GLAMORGAN.

September 21st, 1961. V. F. Parker, Hem House, Rossett, Wrexham, Denbighshire. DENBIGHSHIRE.

September 21st, 1961. W. Holder, The Post Office, Henllys, Cwmbran, Monmouth. MONMOUTHSHIRE.

September 21st, 1961. D. Whitting, Croft Farm, Upton-St.-Leonards, Gloucester. GLOUCESTERSHIRE.

September 21st, 1961. Messrs. H. C. & E. A. Rex, Burley Farm, Bringsty, Worcester. HEREFORD.

September 21st, 1961. J. A. Evans, 15, Ashgrove, Chasetown, Walsall, Staffs. Disease at: Land Off Gas Works Lane, Chasetown. STAFFORDSHIRE.

September 22nd, 1961. R. A. Wintersgill, Springwood Farm, Healey, Masham, Ripon, Yorks. YORK, NORTH RIDING.

September 22nd, 1961. S. Birch, 50, Foxhole Road, St. Thomas, Swansea, Glam. Disease at: Pig Cotes, Kilvey Field, Kilvey Road, Swansea, Glam. COUNTY BOROUGH OF SWANSEA.

September 22nd, 1961. R. M. Britton, Sellake, Uplowman Cross, Tiverton, Devon. DEVONSHIRE.

September 22nd, 1961. D. J. Rees, 1, Harcourt Street, Ebbw Vale, Mon. Disease at: Caswells Field, Ebbw Vale, Mon. MONMOUTHSHIRE.

September 25th, 1961. J. Davies, Longfield Farm, Bromley Green Road, Upper Ruckinge, Ashford, Kent. KENT.

September 25th, 1961. H. Sievers, Coldstream Farm, Croxdale Colliery, Co. Durham. COUNTY DURHAM.

September 25th, 1961. Mrs. E. Ellertson, 24, St. Leger Crescent, St. Thomas, Swansea, Glam. Disease at: Piggeries, Kilvey Hill, Port Tennant, Swansea, Glam. COUNTY BOROUGH OF SWANSEA.

September 25th, 1961. F. Bruton, 44, Tymawr Street, Port Tennant, Swansea, Glam. Disease at: Piggeries, Kilvey Hill, Port Tennant, Swansea. COUNTY BOROUGH OF SWANSEA.

September 25th, 1961. Mrs. E. A. Jones, Penyfodau Fawr Farm, Kingsbridge, Gorseinon, Swansea, Glam. GLAMORGAN.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Cat Among the Pigeons

Sir,—I wonder if you would be good enough to consider some change in the layout of your paper? I am prepared to admit that to an extent the fault may be mine, in that I resent spontaneously any effort to direction from an outside source. Consequently, when my eyes meet two black horizontal lines across a page they instinctively jump the gap. This adds to the interest of life but can be rather shattering. As an example, page 920 of your current issue reads: "The following local authorities sent delegates also: Belfast, Birmingham, Cardiff, Dundee, Glasgow, London, Manchester, Newcastle upon Tyne and Preston. Testicular enlargement in a boar."

I am sure a little research would lead to the discovery of many even more interesting examples.

Yours faithfully,
J. P. PICKERING.

Orchard House,
Priestpopple,
Hexham,
Northumberland.
September 14th, 1961.

[But what can we do? The horizontal lines over which our correspondent hopped as blithely as any young ram were intended as a kind of typographical fence separating one literary pasture from another. Agitation is said to follow the loosing of cats among pigeons; what, then, can be thought of Mr. Pickering loosing a boar (suffering a regrettable disability) among municipal delegates?—*Editor*.]

Downing's "Instructions"

Sir,—Mr. Sherwin A. Hall's most interesting article on J. Downing's "Instructions for the Extracting of Calves, 1797," published in your journal on July 1st, 1961, prompts me to send the enclosed note on a booklet which I found recently in a second-hand book shop. It is entitled "Directions for the Proper Exhibition of Horse Medicines prepared by John Percivall, Assistant Veterinary Surgeon to the Professor of the Veterinary College, 1797."

As Mr. Hall rightly pointed out, early printed works on veterinary medicine dealt in the main with the horse. But, even so, much of what was written at this period on equine matters left a good deal to be desired and was often noted for its lack of caution or feeling for the patient.

Percivall's directions for the use of "Purgative Balls" are given in the following words:—

"PURGATIVE BALLS

"Purgative medicines will be found useful at the commencement of most cases of local inflammation; but where the intestines and lungs are inflamed, the use of purgatives would be imprudent and unsafe. Medicines of this class are very necessary to be given when horses return from grass into stables. While the intestines are distended with green food the animal is incapable of much labour; it is therefore advisable to get rid of their contents by mild purgatives, and which will be found to increase the powers of digestion, improve condition and prevent many diseases.

"If due attention be paid to the horizontal position of the body of horses, and also to the immense length and circumvolutions of the intestines, it is natural to expect that the operation of purgatives must be always slow, nor will it be altogether free from danger, unless the quality and quantity of the medicine be scrupulously attended to. For the bile, or gall (which is the daily purgative in all complicated animals), is of a very mild nature in horses, from there being no gall-bladder; indeed, the stomach and every part of the intestines manifest that their peristaltic motion is naturally very slow.

"If, then, their action be suddenly quickened by a powerful purgative at a time when they are much distended, the intestines cannot readily get rid of their contents, they become highly inflamed and the horse frequently dies.

"This effect is the natural consequence of an over-dose of a stimulating substance laying thirty, forty, or fifty hours, in contact with parts of irritable sensibility. And so various is the constitution of horses, nay, at times so oppositely changeable in the same animal, that some are differently acted on at different periods by the same medicine. The dose, therefore, that will produce salutary effects in one horse, will in another destroy by its excess, and perhaps in a third produce no sensible effect whatever.

"From these circumstances it is evident that no

single dose of purgative medicine can be proper in all horses, or in the same horse at all times.—The common practice is to purge horses by one dose; and I have seen many horses that have died from this cause.

"On this account it is strongly recommended at the college, to divide a purgative ball into two, or three parts, one of which should be given every six and twenty hours, until it purges. By adopting this system of purging horses, it is found that the mildest constitution is not purged too much; and by repeating the dose the proper effect is produced in every case, without the smallest danger; and, as it is the effect only that required attention, whether this takes place from one, two, three, or four repetitions of the medicine, is not of the least importance to the health of the animal.

"Before the horse is purged, it were advisable to feed him but very moderately on bran mashes for a day or two. A diet of this nature will rather tend to empty the bowels, and make the operation of the purgative more safe. We then recommend one third of the purgative ball to be given. Six hours after the first dose is administered the horse should be exercised for two or three hours. Mashes should be continued, and the chill taken from the water. If the first dose produces no sensible effect in twenty-six hours, it should be repeated, the horses should be fed and exercised as before, and if the second dose does not purge, the third should be given.

"When the effect is over, it is better not to give any more purgatives for four or five days. We may then begin cautiously as before; for we are not to expect that the quantity which produced the desired effect in the first instance will always have the same effect when repeated.

"It is usual to give horses neither more or less than three doses of physic. But where the horse is weakly, it is requisite to purge him only once or twice; and in other cases, where there is great plethora, four, or even five times, is not too much."

One cannot help but be impressed by the soundness of this advice, more particularly when one recalls the scathing criticisms that have been heaped on Percivall's teacher, Edward Coleman.

Yours faithfully,
L. P. PUGH.

Department of Veterinary Clinical Studies,
School of Veterinary Medicine,
Madingley Road,
Cambridge.
September 22nd, 1961.

Sir,—May I answer the charge by Professor Francis that there is a "serious misrepresentation of the facts" in my recent article on Downing's Instructions.

In good faith I quoted the reference (Smith, Vol. II, p. 227) and reported on Smith's opinion of Downing, the text of which is as follows:—

"There is no necessity to reproduce Downing's obstetrical operations; they are a classic in our schools, though unacknowledged. When Simonds lectured on obstetrics at the London School over half a century ago he repeated Downing; the diagrams of the College, reproduced by Dobson and Steel, were based on Downing's description of malpresentations, the actual drawings being made from Skelton [*sic*], a man who wrote in the nineteenth century and purloined Downing's work. Downing's name has never been associated with researches in obstetrics; it is gratifying to rescue this Stourbridge practitioner from obscurity. One can only regret that his obstetrical work is associated with the unblushing literary theft of material which is mere dross compared with his own contribution of refined gold."

There can be no doubt that Smith also had a high opinion of Skellett's work if one reads Vol. III, p. 119, of his History. However, the long quotation from this by Professor Francis has several omissions, one of which is of only two sentences. I quote them now in my italics in the original, unaltered context:—

"Modest, unassuming, cool in the presence of great difficulties, kind, humane and gentle in his methods, he stands out as one of the greatest veterinary obstetricians this country has produced.

"*In his Preface he says that the rules of obstetrical practice are not known to any other author. This is unjust to J. Downing (see Vol. II), whose pamphlet on the subject, published in* 1797, *is a revelation.* Skellett may not have seen this, but on comparison there is a resemblance which suggests that he might have drawn on Downing."

If Professor Francis believes that Smith has been unjust to Skellett then critical evidence should be provided to support the thesis. Meanwhile, Smith's is the only published opinion and I do not believe I have misrepresented any facts based on his work.

Yours faithfully,
SHERWIN A. HALL.

Veterinary Investigation Centre,
Ministry of Agriculture, Fisheries and Food,
"Woodthorne,"
Wolverhampton,
Staffs.
September 12th, 1961.

The Behaviour of Cattle in Crushes

Sir,—No doubt others, like myself, have found that where a milking herd has to be tuberculin tested and there is a parlour, the quickest and easiest way is to utilise it as the "crush." It is wise to fill up all the standings except 2, to assist in persuading the animals as they come in to go into one of the standings: the other being occupied by the person carrying out the test. If necessary, it is usually not difficult to remove a bar to facilitate access to the site of injection. If no crush is procurable, the same procedure is followed with bail herds, remembering that the head room is generally restricted; but in all probability I am not alone in having that part well hardened by daily contact with the lid of the luggage boot of my means of transportation!

A common fault in most crushes is that the lower halves of the sides and exit are uncovered. If these are covered the animal is more inclined, as a rule, to keep its head up. Naturally a narrow passage

leading from the collecting yard into the crush is essential, and it is always a good plan to put down plenty of straw in the crush and at the entrance, particularly if the crush has a wooden and slightly elevated floor.

I fully endorse Mr. J. B. White's assessment of the temperament of the different breeds, and nowadays select my testing programme in accordance. There is a distinct advantage in having more than one animal in the crush, so long as the men at the exit, and those holding the bar in between, can be relied upon not to fall asleep!

The younger generation should thank its lucky stars that the subcutaneous test with frequent temperature recording is only of historical interest. Those were the days, to modify a famous utterance, of "toil, tears, profanity, and sweat." It was a common experience to be sent on, during an "interval," to a calving case, or a well-developed uterine prolapse.

Yours faithfully,
J. F. D. TUTT.

"Rothiemurchus,"
St. Cross,
Winchester.
September 23rd, 1961.

Methohexital Sodium in Small-animal Anaesthesia

Sir,—We were very interested to read the article "The Use of Methohexital Sodium in Small-animal Anaesthesia" coinciding with its recent use in this practice. Although our experience is limited to fewer cases than those recorded, we feel that our observations are of value. Recovery has not been so smooth as reported, in that violent paddling movements have occurred. Also, the rate of recovery has been estimated to be about the same as with thiopentone. No muscle tremors were noted during induction when given at the rate we usually adopt for other usual intravenous anaesthetic agents.

However, it is our opinion that methohexital sodium* has several advantages over other common short-acting anaesthetics and hope that more detailed work will be forthcoming.

Yours faithfully,
G. E. JOSS,
J. N. WRIGHT.

Waysmeet,
8, Whitepost Hill,
Redhill,
Surrey.
September 23rd, 1961.

* Brietal sodium: Lilly.

Culturing of Mastitis Milk Samples

Sir,—A few weeks ago you published a paper given by a member of the Veterinary Investigation Service to the East Yorkshire Clinical Club in which the culturing of mastitis milk samples soon after treatment was condemned as most unsatisfactory (Parker, 1961). Unsatisfactory it may be if the whole aim of the exercise is to obtain a regional picture of the incidence of various bacteria in untreated mastitis, but it is certainly not valueless to the general practitioner. The obvious and most practical advantage, of course, is that resistant organisms are still recovered from treated samples. In addition, by relating clinical observations to laboratory findings over a period of years this practice has developed:—

(1) An effective treatment of the acute type of coliform mastitis, and as a result a far better understanding of when, and why, treatment of mastitis will on occasions fail.

(2) A rational and effective policy for the employment of intramammary antibiotics within the herd.

(3) A method of evaluating intramammary antibiotics since the information required is now better understood.

(4) A good picture of local syndromes which would be classified as "sterile mastitis" by the laboratory—and their probable causes.

(5) A picture of the local syndromes when more than one quarter is involved. Only once in 7 years in this practice have organisms not been recovered from such affected untreated quarters in contradistinction to the general mastitis picture.

Yours faithfully,
C. M. GOULD.

12, Landguard Road,
Southampton.
September 23rd, 1961.

Reference

PARKER, W. H. (1961). *Vet. Rec.* **73**, 822.

Humaneness of Cyanide

Sir,—The letter by my old principal, Mr. R. C. G. Hancock, reminded me of happy past days. I was the first assistant of the firm Hancock and Anderson of Uxbridge and Beaconsfield, having been appointed on the death of Mr. Hancock's father, to assist his partner at Uxbridge. My pay was £4 per week and live out, and at the end of 4½ years it had risen to £7 per week with a penny a mile petrol for my car. I paid my own board.

During the whole of the time, Scheele's acid was the method of destroying cats and dogs. I was initiated by the old man's successor (mentioned by Mr. Hancock), who was named Rixon, and who was the handiest man with any animal I have ever met.

I have never liked Scheele's for its obvious danger, and now for dogs prefer liq. strych. by injection, and for cats chloroform by lethal chamber.

Yours faithfully,
L. PITCHER.

66, Coningsby Road,
High Wycombe,
Bucks.
September 20th, 1961.

Heavy Magnesium Bullets and Hypomagnesaemia in Sheep

Sir,—We should like to comment on some of the points raised in a letter by Maguire and Wilson and published in THE RECORD of September 9th.

In our original communication (*Vet. Rec.* **73**, 109) concerning the use of some experimental magnesium bullets in non-pregnant, non-lactating ewes main-

tained on a magnesium-deficient diet composed principally of barley straw, we were able to show that either 2 or 4 bullets maintained plasma magnesium levels at about 1.8 mg. per cent. This level was significantly higher than a control group at 1.2 mg. per cent. over a period of 6 weeks and it took approximately 2 weeks for the magnesium level of the control group to fall from 1.8 to 1.2 mg. per cent. The mean release rate per bullet (as averaged over 16 sheep and 48 bullets) was 73.9 ± 12.5 mg. magnesium per day with a range for individual bullets from 51 to 93 mg.

In a subsequent experiment with non-pregnant ewes on free grazing during a 6-week period in February and March of this year the mean daily release rate (16 sheep and 32 bullets) was 86.5 ± 14.5 mg. During the course of this experiment the control group showed steadily falling plasma magnesium values with several recordings below 1.0 mg. per cent., whereas the group with 2 bullets had no significant reduction and higher magnesium values.

The above determinations of magnesium release rates are much higher than those now quoted by Maguire and Wilson using 2 sheep. In our experience the rate of bullet erosion is determined by the individual sheep in that the release rate of several bullets within the same animal is very uniform.

Maguire and Wilson calculated that to cover a fall of 0.1 mg. per cent. in plasma magnesium for a 50-kg. sheep requires the absorption of 10 mg. of magnesium and concluded that the net metabolisable magnesium of *a single bullet* was between 5 and 8 mg. per day on the assumption that between 17 and 27 per cent. of the daily release rate is "available." They thus concluded that *a single bullet* could not be expected to supply a sufficient quantity of magnesium "to prevent the rapid fall of plasma magnesium levels usually associated with hypomagnesaemic tetany."

Using the same framework of calculation our results would appear to be much more favourable. If the average release rate is 80 mg. magnesium per bullet per day, and even assuming only 25 per cent. availability to the animal, then *2 bullets* would supply 40 mg. of metabolisable magnesium per day. This could be expected to cover a blood plasma reduction of 0.4 mg. per cent. magnesium per day. If a sheep undergoing hypomagnesaemic tetany were to have a fall of, say, 1.0 mg. per cent. of plasma magnesium over a period of 3 days, then this release rate from *2 bullets* might be expected to be in the right order for prevention. We would, however, query the method of assessment of availability as measured by Maguire and Wilson as there are so many complicating factors in such assessments, but at the same time admit that we cannot offer any estimates of our own.

In spite of the above arguments, in trials involving the use of 2 bullets in over 200 lactating sheep on 3 farms in the spring of 1961, the results have been most disappointing. Treated sheep behaved no differently from the controls in that large numbers of both groups developed plasma magnesium values between 0.5 and 1.0 mg. per cent. On the other hand, with a group of 7 of these sheep having such low values on one of the farms, daily drenches of 4,000 mg. of magnesium as calcined magnesite (equivalent to approximately ¼ oz.) also failed to give a sustained response in blood plasma magnesium levels.

Full details of all these experiments are being prepared for publication elsewhere.

Yours faithfully,

R. G. HEMINGWAY,
J. S. S. INGLIS,
N. S. RITCHIE.

Department of Animal Husbandry and Veterinary Preventive Medicine,
University of Glasgow Veterinary Hospital,
Bearsden Road,
Glasgow.

September 18th, 1961.

Mrs. Malaprop up to Date

Sir,—I know you have a feeling for the turn of words and I think the following story will send even your serious readers laughing about their work. On Monday morning a citizen of this town presented his little poodle dog for examination with the history that on the previous day it had been attacked by a mastiff-like hound, described as "about the size of a donkey" and shaken like a rat.

"Sir," he said, "it was 'orrible. My little dog was so terrified he began to execrate."

Yours faithfully,

JOHN PASFIELD.

Shelley House,
Bishopric,
Horsham.

September 20th, 1961.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th August, 1961		3	—	1	25	—	52
Corresponding period in	1960	7	—	—	20	—	42
	1959	5	—	—	29	—	37
	1958	2	—	2	55	—	44
1st January to 15th August, 1961		128	—	103	740	—	742
Corresponding period in	1960	142	—	14	1,377	—	741
	1959	140	—	9	466	—	732
	1958	104	3	113	426	—	865